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PURPURA HÆMORRHAGICA.

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GEORGE ROE LOCKWOOD, M.D.

NEW YORK

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As ordinarily described, purpura hæmorrhagica, or morbus maculosus, is a disease characterized by spontaneous hemorrhages, not only appearing subcutaneously as in simple purpura, but also from the mucous membranes, and more rarely into the serous membranes, internal organs, and joints. This disease was first described by Werlhof, in 1775, and is known as Werlhof's disease.

The patient usually presents prodromal symptoms which may precede the actual onset several days or weeks—malaise, chilly sensations, loss of appetite, and possibly a little rise of temperature being the prodromata most commonly seen. In other cases the disease begins abruptly.

When the disease is fairly developed we have appearing a purpura, the spots varying greatly in size, usually appearing upon the extremities, though they may be generally distributed. There are hemorrhages from various mucous membranes—from the nose, mouth, bronchi, stomach, intestines, and kidneys. These are usually moderate in severity, but may be profuse enough to en-

danger the life of the patient. There are usually constitutional symptoms, chilly feelings (rarely amounting to a rigor at the onset), fever of slight intensity, loss of appetite, and prostration. The gums are normal or slightly swollen, they may bleed, but the teeth are not loosened as in scurvy. The liver and spleen are slightly enlarged; there may be pain and swelling of joints, as in peliosis rheumatica.

Such a disease runs a course of two to four weeks, and tends toward recovery, though relapses are to be expected, and by these relapses the disease may be protracted weeks, months, or years.

In some cases in children the disease manifests itself by purpura, pain and swelling of joints, abdominal pain and tenderness, with tenesmus and bloody stools. It is characteristic of this form of the disease for the patient to suffer from a number of these attacks at short intervals.

Letzerich, in a recent monograph ("Ætiologie und die Kenntniss der Purpura Hæmorrhagica, 1889"), has given us a careful bacteriological study of the disease. He had attended a girl, twenty-five years of age, suffering from Werlhof's disease, in whom a generally distributed purpura, hemorrhages from intestine and kidney, slight fever, prostration, enlargement of liver and spleen were the symptoms noticed. Bacterial examinations carefully performed showed in the purpuric spots the presence of long bacilli capable of growth in gelatine, the pure cultures of which injected into the abdomen of rabbits reproduced the original clinical symptoms in all of twelve cases, and in these the same bacilli were found, identical with those of the pure cultures injected. An examination of the purpuric spots in the rabbits showed dilatation of the capillaries, emigration of white cells, and rupture of the capillary wall allowing of escape of red cells. The capillaries were found filled with the bacilli, with abundant spore growth. (The bacilli and spores had been previously noticed by Petrone, who considered it to be a

mixed infection.) Upon squeezing the section little plugs resembling hyaline casts, containing bacilli, emerged from the capillaries, and these structureless casts he considered as the result of the action of the bacillus or its products upon the fibrino-plastic elements of the blood.

Very remarkable was the appearance of the liver in these rabbits. The organ was regularly enlarged, the portal capillaries were almost occluded by an extraordinary growth of the bacilli. Letzerich considers the liver to be the breeding-place of the bacilli in Werlhof's disease, the liver being to this disease what the spleen is to malarial fever. If he be correct in this view it helps explain both the scattering of the lesions—a bacterial embolism of the capillaries—and also the tendency of the disease to relapse, as well as the periodicity of the relapses seen in some cases. Letzerich himself was attacked by the disease, from which he with difficulty recovered. He attributes his infection to handling his cigar while working at his experiments. The results of other experimenters before the time of Letzerich do not stand the tests of modern requirements and must be excluded.

Aside from these cases in which the disease runs this mild course, we meet with cases in which the same symptoms are present, but in an acute fulminating form. The patient is overwhelmed by the violence and the rapidity of the symptoms, and dies from acute anæmia, internal hemorrhages, or from symptoms of a septic infection. These cases are but briefly referred to, if at all, in the text-books, and it is to these that I would invite your attention. Great care was taken in the selection of these cases to exclude the other forms of hemorrhagic disease to which mention will presently be made.

The first case I have to report is that of a young lady, aged nineteen, living in affluent circumstances. For several years past she had suffered from a moderate degree of anæmia, and at one time was said to have had malarial fever. There was no other previous morbid history. Her father, when a boy, would bleed severely

from slight causes, but further history of hæmophilia could not be obtained. Her illness began on March 7th, though for several days previously she had been ailing from a slight laryngitis.

On March 7th, at 1 A.M., she was seized by a chill and appeared very nervous, the temperature remaining normal. She was given quinine (gr. x.) and remained without symptoms till 12.45 P.M., when she had another chill, the temperature being 101.5° F. during the chill, rising to 103.5° F. within an hour, and then slowly sinking to normal. Shortly after this second chill she was attacked by a nose-bleed, which increased in severity till, by 9 P.M., it had attained an alarming profuseness. Applications of ice, hot nasal douches, insufflation and injection of styptics were vigorously employed; but while each seemed to relieve for a time, the bleeding broke forth with renewed violence after each application. Turpentine and digitalis were begun internally during the night, and continued until noon of the next day.

I saw the patient for the first time at 1 P.M. on March 8th, just twenty-four hours after the acute onset. At that time she was pale and much prostrated. Temperature, 98.4° F., pulse, 130 and dicrotic. She lay in a condition of complete apathy, but her mind was clear when aroused; the expression of the face was that of a typhoid. Upon the trunk, face, and extremities were a number of small purpuric spots. The hemorrhage from the nose still continued, and there was a profuse uterine hemorrhage, although her normal period had ceased two days previously. The gums were normal, there was no fetor to the breath. The collected blood was dark in color and did not coagulate. Careful physical examination showed no evidence of endocarditis or other appreciable disease.

Post-nasal plugging was resorted to, and the anterior nares tightly tamponed and sealed with collodion, after which the hemorrhage was reduced to a slight oozing. During the afternoon she vomited about six ounces of altered blood, which she had apparently swallowed. Small

doses of morphine, digitalis, and brandy were given by mouth and subcutaneously.

By evening her temperature rose to 102.8° F., her pulse varied from 120 to 180, being weak and irregular. Her somnolent condition alternated with periods of restlessness and mild delirium, while new purpuric spots were constantly appearing. Slight oozing came through the nasal plugs; the uterine hemorrhage continued to a moderate extent in spite of careful tamponing.

Dr. L. W. Hotchkiss saw the patient with me at this time, and the question of arterial transfusion was discussed. In view of her hemorrhagic condition, and the extreme difficulty in even checking her bleeding, it was thought advisable not to add a new source of possible hemorrhage by transfusing into an artery, as an equal amount of good might result from saline injections given by rectum and subcutaneously, without danger. Her symptoms, moreover, were more septic than anæmic. Saline solutions were accordingly administered in this way, with an improvement in her symptoms, her pulse becoming slower and stronger, the stupor less profound, and her mind more clear. The improvement, however, was merely temporary. On March 9th, at 9 A.M., the temperature was 104.8° F.; pulse, 148; respirations, 28 to 32; there was no air hunger. At this time she had a black watery offensive movement composed of altered blood. During the afternoon she became more somnolent, the stupor alternating with periods of restlessness and delirium, and new purpuric spots continually appeared. The temperature rose steadily to 106.2° F.; the pulse became extremely rapid, irregular, and feeble; she passed into a condition of coma, in which she died at 2 A.M. on March 10th, two and a half days after the onset of the disease. No autopsy was permitted.

CASE II. (Halbrecht, *V. & H. Jahresber.*, 1884, ii., 268).—Girl, aged ten. Spreading purpura of arms and legs, hemorrhages from bowel, with stupor, headache, and convulsions. Recovery on fourteenth day.

CASE III. (Halbrecht, *V. & H. Jahresber.*, 1884, ii., 267).—Male, thirty years. After attack of urethritis developed mild symptoms of purpura hæmorrhagica; improved, relapsed, and died. Symptoms were, general purpura, bloody diarrhœa, and vomiting of blood. Whole duration three weeks.

CASE IV. (Bourrieff, *Rec. de Mém. de Méd.*, April and May, 1877, quoted *V. & H. Jahresber.*, 1878, ii., 275).—Soldier, aged twenty-two. Purpura, with hemorrhages from kidney, mouth, and lungs. Died in seven hours from hemorrhages into suprarenal capsules.

CASE V. (Vaillard, *V. & H. Jahresber.*, 1878, ii., 276).—Case with hemorrhages from tonsils, epiglottis, and aryepiglottic folds. Death from asphyxia. Details not given.

CASE VI. (Johannsen, *V. & H. Jahresber.*, 1884, ii., 276).—Girl, aged three. Purpura, epistaxis, and hemorrhage from left eye. Recovery ten days.

CASE VII. (Barthelemy, *V. & H. Jahresber.*, 1880, ii., 263).—Male, aged forty-six. Purpura, hemorrhages from kidney, stomach, conjunctiva, mouth, and nose. Death eighth day.

CASE VIII. (Sautin, *V. & H. Jahresber.*, 1880, ii., 263).—Male, aged sixty. Hemorrhages from mouth, nose, ears, bladder, and rectum. Death twenty-first day.

CASE IX. (Wolff, *Berl. Klin. Wochen.*, No. 18, 1879).—Male, aged two years and nine months. Purpura, fever, collapse. Death fifteen hours; hemorrhage into adrenals.

CASE X. (Peszynski, *V. & H.*, 1887, ii., 350).—Male, aged forty-two years. Purpura, blood in urine, stools, and expectoration, fever. Death twenty-first day.

CASE XI. (Appenrodt, *Deutsche Med. Wochen.*, No. 39, 1875).—Child, aged nine months. Purpura, epistaxis, vomiting of blood, blood in stools and urine, apathy, convulsions. Death in four days.

CASE XII. (Woodbury, *Phila. Mea. Times*, September 18, 1886).—Male, aged twenty-three. Purpura, hemorrhages from nose, throat, and kidneys. Death eighth day.

CASE XIII. (Shand, *Lancet*, 1879, ii., p. 79).—Female, aged eight. Purpura, hemorrhages from lungs, bowels, and vagina. Recovered on ninth day.

CASE XIV. (Minich, *Philadelphia Medical Times*, May 8, 1875).—Male, aged seven. Purpura, hemorrhages from nose, mouth, and kidneys, and into parotid gland. Recovered third day.

CASE XV. (Masing, *Vierteljahr. f. Dermat. u. Syph.*, 1887, p. 578). — Male, aged twenty-six. Purpura and epistaxis. Died seventh day.

CASE XVI. (Wagner, *Deutsche Arch. f. Klin. Med.*, Bd. xxxix.).—Male, aged seventeen. Purpura, hemorrhages from mouth and stomach, coma. Death eighth day.

CASE XVII. (Wagner, *loc. cit.*).—Female, aged twenty-eight. Purpura, hemorrhages from gums and uterus for fourteen days, then sudden increase, and died on twentieth day.

Of these 17 cases, 5 were females, 10 were males, and in 2 the sex was not mentioned. The youngest case was one year (the sex not stated). The oldest case, sixty years, a male. The average age of the females was twelve years. The average age of the males was twenty-eight years.

Of the 17 cases 13 died and 4 recovered (3 being females). Shortest duration of fatal cases seven hours; longest twenty-one days; average ten days; average duration of recovered cases nine days.

The autopsies showed hemorrhages under skin, mucous and serous membranes, swelling of the intestinal glands, hemorrhages into the kidneys and muscular tissues, enlargement of the spleen, and in two cases hemorrhage into the adrenals. In one of these latter cases the patient lived but seventeen hours (Bourrieff), in the other fifteen hours (Wolff) after the onset of the disease.

In a second set of cases the course of the disease is characterized by blood-effusion into the brain, death resulting from cerebral hemorrhage.

Case XVIII. (Boucher, *Bull. de la Soc. Anat.*, 1867, No. 43).—Male, aged seventeen, with symptoms of purpura hæmorrhagica. Death from cerebral hemorrhage.

Case XIX. (Hebra, quoted *Gaz. Hébdom.*, 1876, No. 19).—Male, aged nineteen, with general hemorrhages. Death from hemorrhage into the left hemisphere.

Case XX. (Huchard, *Bull. de la Soc. Anat.*, 1874, p. 172).—Male, aged seventeen. General symptoms. Death from cerebral hemorrhage.

Case XXI. (Kurkowski, *V. & H. Jahresber.*, 1885, ii., 493).—Young man. General symptoms. Death fourth day from hemorrhage into left fossa Sylvius, pons, and ventricles.

Case XXII. (Wagner, *Deutsch. Arch. f. Klin. Med.*, Bd. xxxix.).—Girl, aged three, with general symptoms. Death from multiple hemorrhages into the brain.

Case XXIII. (Wagner, *loc. cit.*).—Male, aged thirty-one. General symptoms. Death from subdural and multiple cerebral hemorrhages.

Case XXIV. (Wagner, *loc. cit.*). — Male, fifty-eight years. General symptoms. Death from subdural and multiple cerebral hemorrhages.

Case XXV. (Züelchauer, *Berl. Klin. Woch.*, 1869, No. 17).—Girl, aged two. Diarrhœa and vomiting for two days; then purpura, collapse, and death in a few hours. Hemorrhages in medulla.

Of these 8 cases 6 were males, 4 being seventeen to twenty years; 1 being thirty-one years; 1 being fifty years; 2 were females, two to three years of age respectively.

In a third set of cases when the disease attacks pregnant women it seems to run a rapid and fatal course, causing miscarriage and post-partem hemorrhage.

Case XXVI. (Puech, *Annales de Gynecologie*, xvi., 1887, p. 273).—Woman, aged twenty-one; pregnant six months. Purpura for four days, then purpura spread, developed hemorrhages from nose, gums, kidney, and stomach, with fever and delirium. Miscarriage sixth day, with post-

partum oozing. Died eighth day, four days after sudden increase of symptoms.

Case XXVII. (Stroganow, *Virch. Arch.*, Bd. lxiii., p. 540).—Primipara, aged twenty six; delivery at term. Purpura, collapse, slight fever, post-partum hemorrhage. Death from collapse, twelve hours.

Case XXVIII. (Phillips, *British Medical Journal*, November 13, 1886).—Female, aged twenty-one; pregnant six months. Purpura, colic, epistaxis, delirium, concealed uterine hemorrhage; miscarriage fifth day, post-partum hemorrhage. Death eighth day.

Case XXIX. (Phillips, *loc. cit.*).—Female, aged thirty; five previous normal labors; eight months pregnant. Purpura; miscarriage in few hours. Post-partum hemorrhage and death second day.

Case XXX. (Phillips, *loc. cit.*).—Female, aged thirty-two; seven previous normal labors. In seventh month of pregnancy, purpura, epistaxis, hemorrhage from mouth; miscarriage in three days, with placental hemorrhage. Recovery in two weeks.

When we consider these cases together, we are struck by their similarity to the class of acute infectious diseases. The absence of assignable cause, the rapidity of the onset, the multiplicity and the scattering of the lesions, the enlargement of the liver and spleen, and the constitutional symptoms out of proportion to the local lesions found, seem to prove, by analogy, the assertion that we are here dealing with an acute infection.

If we compare these acute cases with those of the ordinary mild forms of purpura hæmorrhagica, we find the identical symptoms in each, except that in the acuter forms they are more intense and wide-spread, and accompanied by more marked symptoms of a general systemic infection, and it does seem highly probable that in both forms we are dealing with the same disease, caused by the same infection in all essential features, differing only in its intensity of action.

When we are called upon to see patients and diagnose

their disorders, the fact is brought forcibly to our notice that purpura hæmorrhagica is but one of a group of diseases having two essential features in common—tendency to spontaneous hemorrhages and constitutional symptoms. In typical cases each disease of this group may present a characteristic picture, so that we can diagnose it and give to it its particular name. On the other hand, we meet with cases not typical of any one disease but having features common to several; they seem like transitional forms, and we hardly know just under which category to place them. The family resemblance of these diseases is so marked that we cannot close our study of purpura hæmorrhagica without briefly alluding to its relationship to the other hemorrhagic disorders of this group. It is not within the scope of the present paper to enlarge upon these other branches of hemorrhagic disease, but merely to suggest their mutual relationship to each other, as well as to that especial form to which your attention has already been directed.

In order either to affirm or to deny any positive kinship between the different members of this group, we must know in accurate detail the etiology and the pathological processes in each, and unfortunately, until we are able to reason in this way, our conclusions must be simply conjectures and suppositions. Still it would seem well to invite discussion upon this problem, in the hope that by a free expression of opinion we may find how much of fact there is, and how much of theory, in the apparent relationships among the members of this hemorrhagic group.

In the first place, what is the relation between purpura hæmorrhagica and *scurvy?* We find in both symptoms practically identical. A longer prodromal period and a spongy and bleeding condition of the gums are often spoken of as the only points of differential diagnosis between them; but scurvy may begin abruptly, and the gums may be normal, while we may notice in purpura hæmorrhagica long-continued prodromata, with gums swollen and bleeding. It would seem as if the condition of the gums

depended more upon the previous care and cleansing of mouth and teeth, and upon the general condition of the patient, than upon any direct result of the disease. As a matter of fact we call it purpura hæmorrhagica if the patient be well nourished, and scurvy if he be poorly fed and poorly housed.

The older idea of scurvy was that it is a disease of malnutrition from use of improper food, or from absence of fresh vegetables and potassium salts, or from lack of variety in food, or from bad sanitary surroundings. The recent and the more generally accepted idea is that it is an infectious disease of unknown origin. This assumption is based on the fact that scurvy appears frequently in endemics and epidemics, without apparent reason, and on the fact that while the development of the supposed infection is favored by poor food and unhealthy dwellings, these are not essential, and its severity does not seem to be in proportion to the life-degradation of its victims. This view is ably advocated by Koch in the *Deutsche Chirurgie*, Bd. xi., 1889.

If the latter view be correct, why may not the infection be the same as the infection of purpura hæmorrhagica, the symptoms being modified by the unhealthy and poorly nourished condition of the patient, and by the fact that the infection, as a rule, is continued over a longer period of time—a chronic poisoning as it were. If the results of the infection be the same in both instances, it would seem to be a matter of but little consequence whether the actual infecting cause was identically the same in both diseases, whether Letzerich's bacillus was found in scurvy, or some other bacterium, or some ptomaïne found in improper food, capable of inflicting the same mischief. A burn may be caused by an electric wire as well as by a hot iron, but in both instances we call it a burn. Why not simplify, and consider them essentially the same disease, the only difference being that scurvy is more chronic in its infection and that its course is modified by its occurrence in those whose powers of resistance and recupera-

tion are weakened by improper nourishment and unsanitary surroundings.

As regards *simple purpura*, in which we have only subcutaneous and submucous hemorrhages, we find we have to deal with a diversity of causes, in some cases apparent in others obscure and supposititious. Upon studying the effect of these various causes we find great variety in the purpuric symptoms that they excite. In some cases we have only a moderate number of purpuric spots, chiefly seen on the extremities, accompanied by constitutional symptoms of such a mild character that they frequently escape unnoticed; in other cases we have developed a striking exhibition of spreading purpuric lesions with severe general symptoms, such as coma, collapse, and even death; while in other cases we have developed the symptoms of a purpura hæmorrhagica in its most intense form. It is interesting and instructive to note that these various types can proceed from the same cause, acting more intensely upon some patients than in others, either from a maximum of cause on the one hand, or a minimum of resistance on the other.

In the first place we have drug purpura—potassium iodide, chloral, quinine, and salicylic acid being the ordinary drugs during the administration of which a simple purpura is seen. More rarely we have internal and free hemorrhages, and even death resulting, as well as grades of intermediate severity.

Analogous to these cases of drug hemorrhages are the cases which accompany or closely follow severe infectious diseases, as acute yellow atrophy of the liver, snake-bites, typhoid fever, pneumonia, and the exanthemata (so-called black measles, black scarlet fever, hemorrhagic variola). In these cases we have the various grades, from simple purpura up to an acute hemorrhagic purpura. Henoch, for example, describes a case of the severer form as follows (*Berl. Klin. Woch.*, No. 1, 1887): "A child, five years of age, had lobar pneumonia; two days after the crisis developed large spreading ecchymoses of

trunk and extremities, with fever, and death from collapse in twenty-four hours." Such cases are not uncommon, and have been explained by the majority of writers as due to the secondary infection of a Werlhof disease complicating the primary infectious disease from which the patient suffered; purpura hemorrhagica, or as Henoch calls it "fulminating purpura," complicating pneumonia, or acute yellow atrophy, or an exanthem.

If a drug like potassium iodide will disorganize the blood, or render pervious the blood-vessels, so as to allow even a fatal purpura, why may not the poison of these severe infectious diseases produce the same results without supposing an added infection of another disease? It is no argument against this view that the purpuric symptoms frequently appear after the crisis, because we know that a temperature crisis does not always mark the end of the disease (as witness heart-failure in convalescing pneumonia), but only, as Fraenkel[1] has recently demonstrated, the end of the fever-producing quality of the infecting germ.

We may have hemorrhagic disease appearing either as a simple purpura or as purpura hæmorrhagica in profound anæmia, in leucæmia, and in exhausted and cachectic conditions. We do not know whether to attribute its occurrence to blood-changes, or to changes in the walls of the small arteries.

That changes in the blood-vessels can produce hemorrhage is proved by the purpura, bloody sweating, and free hemorrhages, especially of the umbilicus in the newly born infant with congenital syphilitic changes in the arterial walls. Partridge,[2] in an interesting paper read before the Practitioners' Society, describes another form of hemorrhage in newly born infants of non-syphilitic parentage, during the first ten days of life. Such a form occurred in about one per cent. of infants born at the

[1] Path. u. Therap. der Krankheiten des Respirationsapparates, Bd. i., p. 217.

[2] MEDICAL RECORD, August 23, 1890.

Nursery and Child's Hospital and at the Sloane Maternity, with a mortality of sixty to seventy-five per cent. He attributes its cause to the changed condition of functional activities and the altered circulation, allowing a brief interruption of the nutrition of the vessel-walls sufficient for the transudation of their contents.

That simple blood-changes alone will not account for all the cases is also proved by the purpura and hemorrhages seen in cases of malignant endocarditis, where we suppose capillary embolism of vegetation fragments to be the exciting cause.

Fagge[1] has reported a set of cases where purpura, hemorrhages from mucous membranes, rheumatic pains, and fever were noticed in patients suffering from multiple sarcomata. It is hard to say whether these result from the profound alteration of the blood due to the malignant disease, or whether detached sarcomatous fragments become lodged in the capillary vessels, and cause their softening after the manner of emboli.

Besides these changes in the arterial walls, it is supposed that a local vaso-motor relaxation, or an enfeeblement of the wall following fright, severe emotions, or due to hysterical or hypnotic influence, may be followed by purpura and hemorrhage. Such cases are frequently reported in literature. Weir Mitchell, in discussing these cases, says:[2] "We are justified in asking whether neural conditions may not be capable of directly enfeebling the vessel-walls so as to cause them to give way under arterial pressure, or to permit of such relaxation as shall enable the globules to penetrate these parts in the manner of Waller and Cohnheim?" Just how far this weakening from neural influence can be applied to other forms of hemorrhages, the nerves being affected by organized or unorganized blood-poisoning, we cannot say.

We now come lastly, to two forms of purpura to which no apparent cause can be attributed. We call these *pur-*

[1] Guy's Hospital Reports, vol. xxv.
[2] American Journal of the Medical Sciences, July, 1869.

pura simplex and *purpura rheumatica*, or peliosis rheumatica. In both we have simple purpura, but in the latter form associated with pain and swelling of the joints. They have been formerly considered as distinct diseases, but of late effort has been made to associate them, it being urged that peliosis rheumatica is that form of purpura which occurs in rheumatic subjects, the joint symptoms being due to the associated rheumatism, or that it is a severer form of purpura in which hemorrhages occur in or around the joints. That the joints may be so involved in purpuric diseases admits of scarcely any doubt. The joint symptoms are seen in simple Werlhof's disease, in scurvy, in Fagge's cases with multiple sarcomata, and in many other varieties of hemorrhagic dyscrasiæ. If this be so, why consider them as separate diseases? Is it not justifiable to call purpura rheumatica an intenser form of purpura with joint lesions of a hemorrhagic nature?

If Werlhof's disease be due to an acute infection, may not some of these cases at least both of simple purpura and peliosis, be cases of a lighter form of the same infection? To support this view may be cited cases such as the following of Hervé's, which seem to occupy a middle ground between the two conditions. Hervé (quoted in "Keating's Encycl.," ii., p. 83) reports an infant aged three months, who for twenty-four hours had suffered from restlessness, malaise, pallor, rapid pulse and respiration, with mucous râles in the chest. No gastro-intestinal symptoms. At the end of twenty-four hours developed spreading purpura of legs, thighs, and abdomen, with coldness of skin and some œdema, without free hemorrhages from mucous membrane, and died in ten hours.

In summing up, the points to which discussion is invited are:

1. Werlhof's disease is probably infectious in origin, the exact infection not having been absolutely proven, though probably it is the bacillus described by Letzerich.

2. We meet acute cases of this infection, in which

death results from acute anæmia, from internal hemorhages or from sepsis.

3. Purpura simplex and purpura rheumatica are probably types of different grades of the same infection, and this infection may be the same as that of Werlhof's disease.

4. Scurvy, if proven an infectious disease, may be really Werlhof's disease modified by the surroundings and poor condition of the patient, and also by the possibility of the infection being more chronic.

5. Drug purpuras, anæmic and cachectic purpuras, purpuras in exanthemata and other infectious diseases, purpuras in newly born, in endocarditis and multiple sarcomata, as well as those of neural origin, may present all grades of severity; that we can in each determine a cause, though we do not know exactly how the symptoms are produced by this cause, whether by blood-changes or vessel-changes, or from nervous causes; but these purpuras are symptomatic and not essential, and should not be classed with purpura hæmorrhagica, or Werlhof's disease, until we are more enlightened upon this subject.